

Subtotal Resection of the Head of the Pancreas Combined with Ductal Obliteration of the Distal Pancreas in Chronic Pancreatitis

R. P. KERREMANS* F. M. PENNINGCKX* J. FEVERY†
J. DE GROOTE†

Subtotal resection of the head of the pancreas combined with duct obliteration of the distal pancreas by prolamine was performed in 12 selected patients who had chronic alcohol-induced pancreatitis with most destruction in the proximal pancreas. The main indication for operation was intractable pain. There was no postoperative mortality but morbidity was high when no pancreaticojejunostomy was constructed. After a follow-up period of 32 months, lasting pain relief was obtained in 10 patients; pseudocyst formation occurred in three patients; calcification of the distal pancreas, absent before operation, was demonstrated in four of six patients; six of 11 nondiabetic patients became hyperglycemic either abruptly (1 patient) or progressively (5 patients); quality of life improved in most patients. This procedure preserves the stomach, duodenum, spleen, distal pancreas and common bile duct if possible. However, pancreatic ductal obliteration with prolamine does not prevent relapses of chronic pancreatitis.

SURGERY PLAYS AN IMPORTANT ROLE in the management of patients with chronic alcohol-induced pancreatitis (CAIP). About 50% of them will have to have surgery sooner or later.¹⁻³ In the absence of large pseudocysts, pseudoaneurysm, biliary or duodenal obstruction, surgery for chronic pancreatitis is done mainly to relieve medically intractable pain with the lowest possible complication rate. Surgical interruption of nerve pain pathways seems to be very logical but has largely been abandoned. Celiac plexus block was also found to be unhelpful in too many patients.⁴ Indeed, the exact mechanisms of chronic pancreatic pain are still uncertain. Operative strategy for pain relief is now tailored to the morphologic alterations of the pancreatic gland as can be imaged by ultrasound scanning, computer tomography (CT), and endoscopic retrograde cholangiopancreatography (ERCP). Patients with dilated major ducts should

From the Departments of Gastroenterological Surgery and Internal Medicine,† University Clinic Gasthuisberg, Catholic University of Leuven, Leuven, Belgium*

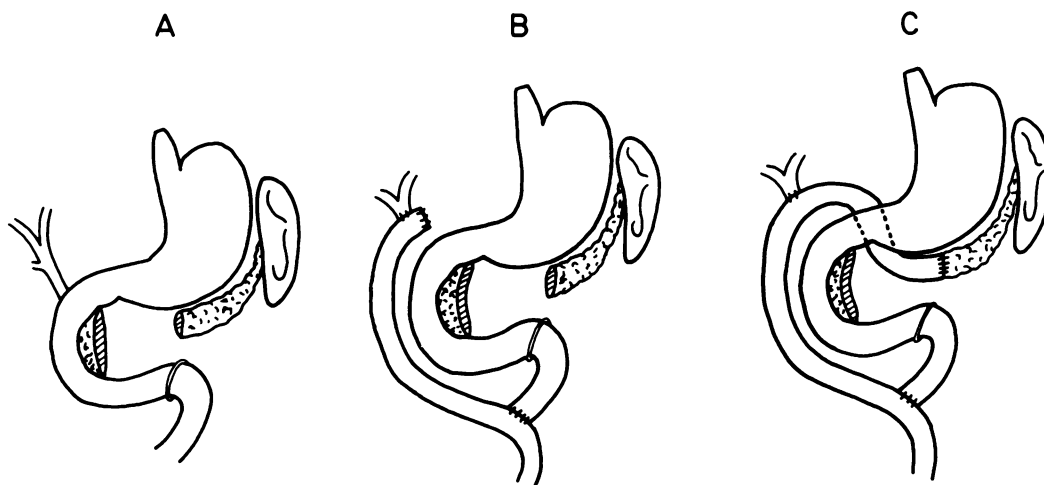
be treated by duct drainage procedures.⁵⁻⁸ Patients with severe pancreatic fibrosis and small- or normal-sized major pancreatic ducts are candidates for pancreatic resection: 40-80% subtotal pancreatectomy for disease localized to the body and tail, pancreaticoduodenectomy for lesions in the head, and 80-95% near total pancreatectomy or even total pancreatectomy for diffuse pancreatic fibrosis.⁹⁻¹¹ The main drawbacks of extended distal resections are the higher early and late morbidity and mortality mostly related to the endocrine insufficiency. To save the endocrine function of the pancreatic tail, segmental pancreatic autotransplantation to the femoral vessels and intrahepatic islet autotransplantation have been suggested. Autotransplantation of the complete denervated pancreatic tail is an attractive technique but it sacrifices the spleen and makes the procedure even more complicated,¹²⁻¹⁴ whereas the achievement of glucose homeostasis with islet cell transplantation has not been encouraging until now.^{7,15,16}

As the disease is found to be concentrated in the head of the pancreas more frequently than was noted before the availability of ERCP, few 80-95% distal pancreatectomies and more pancreaticoduodenectomies (Whipple's procedure) are being performed.¹⁷ To simplify the resection and to prevent postgastrectomy symptoms after Whipple's resection, duodenectomy with pylorus preservation¹⁸⁻²¹ as well as duodenum-preserving subtotal resection of the head of the pancreas^{22,23} have been applied in patients with CAIP. Obliteration of the pancreatic duct by prolamine at Whipple's procedure has been proposed to interrupt the chronic inflammatory process in the remaining pancreatic tail, preserving at the same time its endocrine function.^{17,18,24}

Reprint requests: R. P. Kerremans, M.D., Department of Gastroenterological Surgery, University Clinic Gasthuisberg, Herestraat 49, B-3000 Leuven, Belgium.

Submitted for publication: September 18, 1986.

FIGS. 1A–C. Subtotal pancreatic head resection without any anastomosis (A), with biliodigestive anastomosis (B), with biliodigestive and pancreaticodigestive anastomosis (C).



In a very select group of patients with major destruction by CAIP in the pancreatic head and/or body and only minor lesions in the pancreatic tail, we have performed a subtotal resection of the pancreatic head as in the 95% subtotal pancreatectomy but with preservation of the left pancreas and the spleen in combination with obliteration of the left pancreatic duct with prolamine. The aim of this combined procedure was not only to obtain lasting pain relief but also to save the distal pancreas, spleen, stomach, duodenum, and, if possible, the common bile duct to prevent recurrences of pancreatitis in the distal pancreas, preserving at the same time its endocrine function. The results obtained after 6–54 months of follow-up are reported.

Materials and Methods

Twelve patients with CAIP were selected for subtotal pancreatic head resection and left pancreatic duct obliteration by prolamine injection. All but two patients stopped alcohol intake at least 3 months before surgery. All patients were men ranging in age from 34–48 years, with a mean age of 40.8 years (± 4.9 years SD; minimum 35 years and maximum 48 years). Indications for operation included severe pain (12 patients), obstructive jaundice (2 patients), suspicion of pancreatic malignancy (2 patients) and pseudoaneurysm of the left gastric artery (1 patient). Duration of pain ranged from 1–9 years (mean: 3.9 ± 2.6 years SD) and was more than 3 years for seven patients. Eight patients were addicted to narcotic analgesics. Preoperative symptoms also included weight loss (11 patients: 15.5 ± 8.0 kg), gastroduodenal complaints (7 patients), steatorrhea (5 patients with fecal fat greater than 7 g/day). One patient had diabetes mellitus, and the oral glucose tolerance test was abnormal in seven patients.

The morphologic features of the diseased pancreas were evaluated by CT scan, ultrasonography, and ERCP or intraoperative pancreatography in all patients. Angiography

was performed in seven patients and showed abnormalities in four of them: pseudoaneurysm in one patient and compression or displacement of the superior mesenteric artery in three patients. On CT scan the head of the pancreas was grossly enlarged in all patients and presented one or more cystic cavities in seven patients. One patient showed a pseudocyst in the body of the pancreas in contiguity with a pseudoaneurysm of the left gastric artery. Pseudocysts in the distal pancreas were never visualized in this patient series as this was considered a contraindication to the surgical approach reported here. Eleven patients had pancreatic calcifications: six diffusely and five only in the pancreatic head. Ultrasonography did not provide supplementary information, and adequate visualization of the distal pancreas was frequently impossible. The main pancreatic duct could not be catheterized in four patients. Pancreatography was abnormal in the other eight patients, including segmental obstruction, stricture in the head or body with no or moderate distal dilatation in eight patients, and filling of pseudocysts (less than 4 cm in diameter) in two patients. Cholangiography was obtained in 10 patients and showed common bile duct stenosis in its lower part in six patients.

The operative procedure starts with ventral dissection of the pancreas. In instances of suspected malignancy, biopsy specimens are obtained for frozen section examination. A cholecystectomy and perioperative cholangiography are performed. After choledochotomy, a Bakès dilator is introduced in the common bile duct and passed through the papilla. The resection begins with tunneling and division of the pancreas at the border between body and tail leaving intact the splenic vessels. The subtotal resection is executed from the cut edge towards the prepapillary common bile duct over the portomesenteric vein. A cuff of 5–10 mm of pancreas lining the lesser curvature of the duodenum is retained (Fig. 1A). If the lower bile duct is injured due to peripancreatic fibrosis, a Roux-en-Y hepaticojejunostomy is constructed (7 pa-

tients, Fig. 1B), otherwise the choledochotomy is closed on a T-tube. The main and secondary ducts of the preserved distal pancreas are obliterated by prolamine injection (Ethibloc®, Ethicon GmbH, D-2000 Norderstedt, West Germany) under radioscopic control. After injection, the Wirsung duct is ligated. The jejunal loop used for the biliodigestive anastomosis can also be sutured to the cut edge of the pancreatic tail to drain pancreatic juice eventually leaking from the pancreatic section surface (5 patients, Fig. 1C). The space between the left pancreas and the duodenal rim of pancreatic tissue is drained.

All patients were checked at least once a year. Data relevant to pain behavior, abstinence from alcohol, use of analgesics, work behavior, fat malabsorption, endocrine pancreatic function, and liver function were collected. Patients with pain relapses or diabetes mellitus that was difficult to manage were readmitted to our wards. A CT scan was performed between 2 and 3 months after operation in all patients; further CT scans were performed in 10 patients usually at 1-year intervals.

Results

The results were analyzed according to five criteria: (1) relief of pain; (2) incidence of calcification and pseudocyst formation in the distal pancreas; (3) evolution of pancreatic endocrine function; (4) operative morbidity and mortality; and (5) quality of life as determined by withdrawal from analgesics and alcohol, evolution of body weight, and work behavior. The actual evaluation of our patients is listed in Table 1.

Relief of Pain

Prompt pain relief occurred in all patients after operation. Pancreatic pain relapse was observed after 4 months in one patient; at that time the diagnosis of recurrent pancreatitis with pseudocyst formation in the distal pancreas and duodenitis was made. This patient died 10 months after operation; at necropsy, acute ischemic necrosis of the small intestine and right hemicolon, atherosclerosis with stenosis of the celiac trunk, superior and inferior mesenteric arteries (critical stenosis of SMA), and chronic calcifying pancreatitis of the distal pancreas with pseudocyst formation were found. Pain relapse occurred after 22 months in another patient who never stopped alcohol intake. Both patients had an external pancreatic fistula in the immediate postoperative period closing after 5 and 7 months, respectively. It can be estimated by actuarial techniques that 80% of the patients remain pain free at 2 and 5 years follow-up.

It has to be mentioned that two patients had pain relapses due to ulcer disease: one antral and one bulbar ulcer. The latter was successfully treated with cimetidine; the antral ulcer was present before operation but a stoma

TABLE 1. *Actual Results*

Patient No.	Follow-up (mo)	Pain Relief	Pseudocyst	Diabetes	Work
1	54	Y	Y	Y	N
2	51	Y	N	Y	N
3	50	Y	N	Y	N
4	49	Y	N	Y	Y
5	42	Y	N	N	Y
6	33	N	Y	N	Y
7	29	Y	N	N	N
8	24	Y	N	Y	N
9	23	Y	N	Y	Y
10*	10	N	Y	N	N
11	10	Y	N	N	Y
12	6	Y	N	Y†	Y

* Died of intestinal ischemia.

† Was diabetic before operation.

ulcer developed after truncal vagotomy and antrectomy performed 30 months after the partial pancreas resection.

Incidence of Calcification and Pseudocyst Formation in the Distal Pancreas

Calcifications could not be demonstrated in the distal pancreas of six patients by plain abdominal roentgenograms or CT scan performed just before surgery. In four of these six patients, progressive calcification of the pancreatic tail could be demonstrated in the first postoperative year in two patients and in the second year in the other two patients.

No patient had even small pseudocysts in the distal pancreas before operation. The development of one or more pseudocysts within or alongside the pancreatic tail was demonstrated by CT scan in three patients in the first, second, and fifth postoperative year, respectively.

Evolution of Pancreatic Endocrine Function

Six of 11 nondiabetic patients developed hyperglycemia immediately after operation (1 patient), in the second half-year (2 patients), or in the second postoperative year (3 patients). Currently, all of them are insulin-dependent. The endocrinologic evolution is independent of the preoperative functional state: three of four patients with normal preoperative oral glucose tolerance test (OGTT) developed diabetes and three of seven patients with pathologic OGTT before operation. Before operation, five nondiabetic patients had calcifications in the distal pancreas; two of them are diabetic now. Of the six nondiabetic patients without calcifications in the distal pancreas, four became diabetic, three of them also had calcifications in the pancreatic tail. The endocrine pancreatic insufficiency developed abruptly after surgery in only one patient. A progressive deterioration of glucose homeostasis and

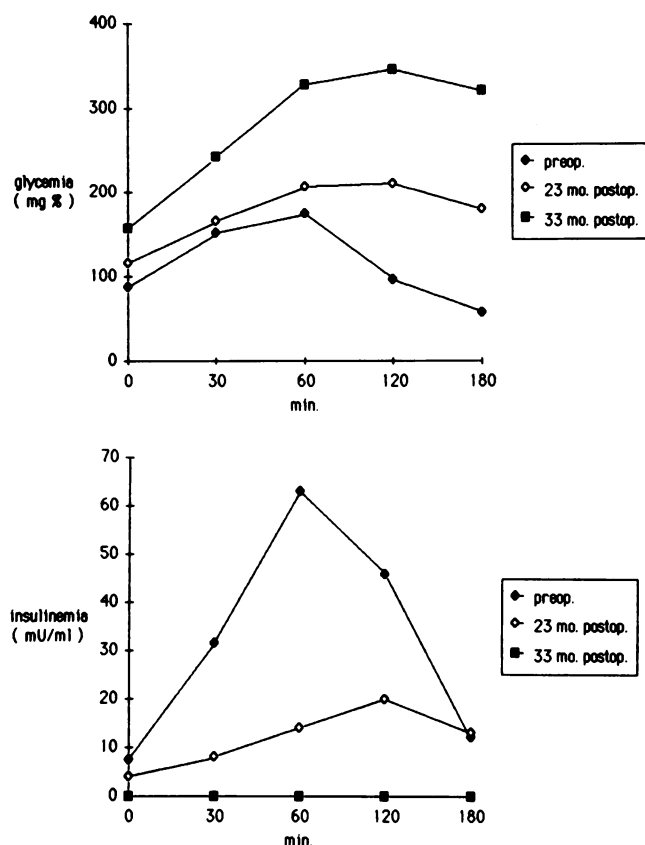


FIG. 2. Glucose homeostasis and insulin secretion in a patient after oral glucose tolerance tests (10 g glucose/10 kg body weight) performed before and at several times after subtotal pancreatic head resection combined with obliteration of the distal pancreatic duct by prolamine.

glandular insulin and C-peptide exhaustion could be demonstrated in the other five patients (Fig. 2).

Postoperative Morbidity and Mortality

There is no early postoperative mortality. One patient died of intestinal ischemia 10 months after operation.

Three of 7 patients in whom no pancreaticojejunostomy was performed had an external pancreatic fistula originating from the distal pancreas; these fistulae closed after 1, 5, and 17 months, respectively; a pseudocyst developed in two patients. An external fistula communicating with the peripapillary region of the duodenum developed in two patients; these fistulae closed within 17 and 30 days, respectively. One patient had to be reoperated on at day 5 because of a perforated duodenal ulcer that was simply closed.

The main duration of hospital stay was 21 days (± 6.7 days SD).

Quality of Life

One patient never stopped intake of alcohol. Of the 11 patients who withdrew from alcohol before operation, two

relapsed. No patient remained addicted to narcotic analgesics after operation, although two patients were taking analgesics because of recurrent pancreatic pain.

Six patients were gainfully employed at the time of this study; between them are four of nine patients who were not working before operation. Although exocrine pancreatic insufficiency was surgically induced in all patients, five patients were not using enzyme substitution without subjective adverse effect; only one of these five patients had a pancreaticojejunostomy. At the time of this analysis only five patients had gained some weight; however, no one reached his ideal body weight.

Discussion

It is believed that progressive loss of exocrine function occurring over the years together with calcification of the pancreas is related to late pain relief.²⁵ Destruction or hastening loss of exocrine function while preserving endocrine function has been the aim of some types of surgical management: duct ligation and obliteration. Ligation of the main duct has been used with variable success in the management of pain from chronic pancreatitis.²⁶⁻³⁰ However, it has been shown to induce progressive endocrine dysfunction in animals³¹ and in humans.³⁰ More recently, the injection of prolamine or cyanoacrylate glue into the ductal system has been used.^{32,33} Excellent early results have been reported after pancreaticoduodenectomy with or without pylorus preservation, combined with pancreatic duct obliteration by prolamine,^{24,32} whereas the experience with surgical or endoscopic obliteration of the pancreatic duct by acrylate^{33,34} or prolamine^{24,35,37} without resection has not been so favorable. Indeed, complete obliteration of the duct system over its whole length may not be feasible in chronic pancreatitis because of preexisting ductal lesions.

In the current series of patients with CAIP and most destruction in the head and body of the pancreas, we combined a subtotal pancreatic head resection with ductal obliteration of the distal pancreas by prolamine injection. An identical surgical approach *without* pancreatic duct obliteration has been developed independently by others.²³ Our results confirm that it is possible to save the distal pancreas, spleen, stomach, and duodenum in chronic pancreatitis, while prompt pain relief was obtained after operation in all patients so treated. Due to peripancreatic fibrosis it has not been possible to save the integrity of the common bile duct in seven of eight patients with more or less distal bile duct entrapment. Although prophylactic bypass of the common duct at the time of surgery is not warranted,³⁸ it has been emphasized that biliary cirrhosis may develop insidiously even in the absence of jaundice.³⁹⁻⁴¹ Up to now, no common bile duct complications have been observed either biochemically or by radiologic imaging in any of our patients.

Although the combined procedure proposed in this report is complex, it provided lasting pain relief in almost 80% of patients with pain that was qualified as medically intractable at the time of operation since 4 years on average. In such patients, further waiting during an undetermined time to get spontaneous pain relief in some^{2,42} was not considered a human alternative. However, it has to be admitted that postoperative lasting pain relief is probably not due to the operation alone but also to alcohol abstinence in most of our patients. Although postoperative progression of the disease has been observed after alcohol abstinence and derivative or resectional surgery^{3,43} withdrawal from alcohol has been proven to be a major factor in relation to continued postoperative pain relief.^{1,25,44-46}

We have added duct obliteration of the distal pancreas by prolamine injection to the subtotal resection of the head of the pancreas to prevent recurrences of pancreatitis in the pancreatic tail, preserving at the same time its endocrine function. Lasting pain relief is not an adequate parameter of the evolution of the disease as successful relief of pain does not preclude progression of the chronic pancreatitis process.⁴⁷ Thus, the endocrine function and morphology of the remaining pancreas were regularly checked in all our patients. The results indicate that duct obliteration with prolamine does not prevent relapses or progression of chronic pancreatitis in the distal preserved pancreas: (1) exocrine secretion is not always blocked since external pancreatic fistulae originating from the distal pancreas and pseudocyst formation were observed; (2) calcification of the tail was not prevented but may be related to the inflammation and fibrosis provoked by prolamine; and (3) progressive endocrine pancreatic insufficiency has been documented. As these unfavorable results might be related to technical aspects of the procedure, intraoperative radioscopy and radiography demonstrated filling of the main and secondary ducts of the distal pancreas with prolamine and the main duct has been ligated at the section surface after injection of prolamine in all patients. A comparison between our early results at 1 year of follow-up⁴⁸ and these late results illustrate that progressive endocrine pancreatic destruction is not avoided by ductal obliteration. Insignificant differences in endocrine function between patients with and without pancreatic ductal obliteration followed during 2 years have also been reported by others.⁴⁹ Thus, it may be concluded that the endocrine function is not sacrificed by the procedure itself, although it has not been demonstrated unequivocally that prolamine injection does no harm to the islet cells.⁵⁰⁻⁵²

An additional benefit of prolamine pancreatic duct obliteration has been that the incidence of anastomotic leaks at the pancreaticojejunostomy was reduced, contributing to a significantly decreased postoperative mortality.^{18,49} However, this procedure does not permit omis-

sion of pancreaticojejunostomy construction with safety²⁰ as is confirmed by the occurrence of pancreatic fistulae in three of seven of our patients in those circumstances. This demonstrates that it is not always, if ever, possible to completely stop secretion in the residual exocrine pancreatic tissue. Although acinar atrophy has been documented experimentally in normal pancreases of dogs after pancreatic duct obliteration with prolamine,⁵⁰ the continued presence of pancreas-isoamylase and trypsin in the sera of patients with chronic pancreatitis and distal duct occlusion indicates residual exocrine secretion.²⁴ Nevertheless, we should pursue our efforts to stop the inflammatory process in the remaining pancreas and to preserve its endocrine function as considerable data support the concept that there is continued deterioration once pathologic changes of chronic pancreatitis have occurred.

The procedure proposed here permits preservation of the stomach, duodenum, distal pancreas, spleen, and, if possible, also the common bile duct, while it provides lasting pain relief and an improved quality of life in the majority of patients. Exocrine insufficiency is easily treated with oral pancreatic enzyme replacement therapy and reduction of daily fat intake. In our opinion this new surgical approach is indicated in patients with intractable pancreatic pain after alcohol withdrawal during at least 6 months due to fibrocalcific duct obstruction with or without cyst formation in the proximal pancreas and a small or normal-sized distal pancreatic duct. This is not a rare situation as obstruction due to chronic pancreatitis occurs most frequently in the head of the pancreas. Major ductal strictures and cyst formation in the distal pancreas are to be considered contraindications to this approach as well as portal hypertension, previous pancreaticojejunostomy, and the presence of a pseudoaneurysm of which the afferent blood supply would not be interrupted by the resection.

References

1. Scuro LA, Piubello VW, Micciolo R, et al. Evolution of pain in chronic relapsing pancreatitis: a study of operated and nonoperated patients. *Am J Gastroenterol* 1983; 78:495-501.
2. Ammann RW, Akovbiantz A, Largiader F, Schueler G. Course and outcome of chronic pancreatitis. *Gastroenterology* 1984; 86:820-828.
3. Nogueira CED, Dani R. Evaluation of the surgical treatment of chronic calcifying pancreatitis. *Surg Gynecol Obstet* 1985; 161: 117-118.
4. Leung JW, Bowen-Wright M, Aveling W, et al. Coeliac plexus block for pain in pancreatic cancer and chronic pancreatitis. *Br J Surg* 1983; 70:730-732.
5. Jordan GL, Strug BS, Crowder WE. Current status of pancreaticojejunostomy in the management of chronic pancreatitis. *Am J Surg* 1977; 133:46-51.
6. Warshaw AL, Popp JW, Schapiro RH. Long term patency, pancreatic function and pain relief after lateral pancreaticojejunostomy for chronic pancreatitis. *Gastroenterology* 1980; 79:289-292.
7. Morrow CE, Cohen JI, Sutherland DER, Najarian JS. Chronic pancreatitis: long-term surgical results of pancreatic duct drainage,

- pancreatic resection, and near-total pancreatectomy and islet autotransplantation. *Surgery* 1984; 96:608-616.
8. Prinz RA, Aranha GV, Greenlee HB. Redrainage of the pancreatic duct in chronic pancreatitis. *Am J Surg* 1986; 151:150-156.
 9. Frey CF, Child CG, Fry W. Pancreatectomy for chronic pancreatitis. *Ann Surg* 1976; 184:403.
 10. Taylor RH, Bagley FH, Braasch JW, Warren KW. Ductal drainage or resection for chronic pancreatitis. *Am J Surg* 1981; 141:28-33.
 11. Traverso LW, Tompkins RK, Urrea PT, Longmire WP. Surgical treatment of chronic pancreatitis. *Ann Surg* 1979; 190:312-317.
 12. Hogle HH, Reemtsma K. Pancreatic autotransplantation following resection. *Surgery* 1978; 83:359-360.
 13. Tossatti E, Valente U, Campisi C, et al. Segmental pancreas autotransplantation in man following total or near total pancreatectomy for serious recurrent chronic pancreatitis. *Transplant Proc* 1980; 12(suppl 2):15-18.
 14. Rossi RL, Braasch JW, Nugent FW, et al. Segmental pancreatic autotransplantation for chronic pancreatitis. *Am J Surg* 1983; 145:437-442.
 15. Cameron JL, Mehigan DG, Broe PJ, Zuidema GD. Distal pancreatectomy and islet autotransplantation for chronic pancreatitis. *Ann Surg* 1981; 193:312-317.
 16. Sutherland DER. Pancreas and islet transplant registry data. *World J Surg* 1984; 8:270-275.
 17. Gall FP, Mühe E, Gebhardt C. Results of partial and total pancreaticoduodenectomy in 117 patients with chronic pancreatitis. *World J Surg* 1981; 5:269-275.
 18. Gall FP, Mühe E, Gebhardt C. Etude comparative de 117 duodéno-pancréatectomies subtotaux et totales pour pancréatite chronique. *Chirurgie* 1979; 105:187-192.
 19. Traverso LW, Longmire WP. Preservation of the pylorus in pancreaticoduodenectomy. *Surg Gynecol Obstet* 1978; 146:959-962.
 20. Flautner L, Tihanyi T, Szécsényi A. Pancreatogastrostomy: an ideal complement to pancreatic head resection with preservation of the pylorus in the treatment of chronic pancreatitis. *Am J Surg* 1985; 150:608-611.
 21. Braasch JW, Rossi RL. Pyloric preservation with the Whipple procedure. *Surg Clin North Am* 1985; 65:263-271.
 22. Beger HG, Witte C, Kraas E, Bittner R. Erfahrung mit einer das Duodenum erhaltenden Pankreaskopfresektion bei chronischer Pankreatitis. *Chirurgie* 1980; 51:303-309.
 23. Beger HG, Krantzberger W, Bittner R, et al. Duodenum-preserving resection of the head of the pancreas in patients with severe chronic pancreatitis. *Surgery* 1985; 97:467-473.
 24. Schneider MU, Lux G, Gebhardt Ch, et al. Therapeutische Pankreasgangocclusion bei chronischer Pankreatitis: klinische, exokrine und endokrine Konsequenzen bei 12monatiger Nachbeobachtung. *Langenbecks Arch Chir* 1985; 363:149-163.
 25. Ammann RW, Largiader F, Akovbiantz A. Pain relief by surgery in chronic pancreatitis? *Scand J Gastroenterol* 1979; 14:209-215.
 26. Cannon JA. Experience with ligation of the pancreatic ducts in the treatment of chronic relapsing pancreatitis. *Am J Surg* 1955; 90:266-280.
 27. Madding GF, Kennedy PA, McLaughlin B. Obstruction of the pancreatic duct by ligature in the treatment of pancreatitis. *Ann Surg* 1967; 165:56-60.
 28. Madding GF, Kennedy PA. Chronic alcoholic pancreatitis. *Am J Surg* 1973; 125:538-541.
 29. Hoffmann E, Usmiani J, Gebhardt Ch. Die Ausschaltung der exokrinen Funktion des Pankreas als Behandlungskonzept der chronischen Pankreatitis. *Dtsch Med Wochenschr* 1977; 102:392-395.
 30. Hutson DG, Levi JU, Livingstone A, Zeppa R. Pancreatic duct ligation in therapy of chronic pancreatitis. *Am Surg* 1979; 45:449-452.
 31. Idezuki Y, Goetz FC, Lillehei RC. Late effect of pancreatic duct ligation on beta cell function. *Am J Surg* 1969; 117:33-39.
 32. Gall FP, Gebhardt Ch. Ein neues Konzept in der Chirurgie der chronischen Pankreatitis. *Dtsch Med Wochenschr* 1979; 28:1003-1006.
 33. Little JM, Hogg J, Stephen M. Duct obliteration with an acrylate glue for treatment of chronic alcoholic pancreatitis. *Lancet* 1979; 2:557-558.
 34. Ward WF. Intrapaneatic pseudocyst formation: a complication of acrylate pancreatic duct obliteration. *Aust NZ J Surg* 1981; 51:494-496.
 35. Van Bastelaere W, Derom F. Nieuwe trend in de heekundige behandeling van chronische pancreatitis. *Acta Chir Belg* 1983; 83:60-64.
 36. Flautner L, Papp J, Tihanyi T, et al. Endoskopische Occlusionsbehandlung bei Patienten mit chronischer Pankreatitis. *Chirurg* 1985; 56:36-40.
 37. Pap A, Flautner L, Tihanyi T, et al. Pancreatic function after endoscopic and surgical occlusion of the pancreatic duct in patients with chronic pancreatitis. *Endoscopy* 1985; 17:60-63.
 38. Bradley EL. Parapancreatic biliary and intestinal obstruction in chronic obstructive pancreatitis. *Am J Surg* 1986; 151:256-258.
 39. Afroudakis A, Kaplowitz N. Liver histopathology in chronic common bile duct stenosis due to chronic alcoholic pancreatitis. *Hepatology* 1981; 1:65-72.
 40. Eckhauser FE, Knol JA, Strodel WE, et al. Common bile duct strictures associated with chronic pancreatitis. *Am Surg* 1983; 49:350-358.
 41. Brinton MH, Pellegrini CA, Stein SF, Way LW. Surgical treatment of chronic pancreatitis. *Am J Surg* 1984; 148:754-759.
 42. Kondo T, Hayakawa T, Noda A, et al. Follow-up study of chronic pancreatitis. *Gastroenterol Jpn* 1981; 16:46-53.
 43. Adloff M, Ollier JC, Sehloegel M. Les opérations de drainage dans le traitement des pancréatites chroniques. A propos de 81 cas. *Chirurgie* 1985; 111:371-377.
 44. White TT, Keith RG. Long term follow-up study of fifty patients with pancreaticojejunostomy. *Surg Gynecol Obstet* 1973; 136:353-358.
 45. Greenlee HB. The role of surgery for chronic pancreatitis and its complications. *Surg Annu* 1983; 15:283-305.
 46. Ink O, Labayle D, Buffet C, et al. Pancréatite chronique alcoolique: relations de la douleur avec le sevrage et la chirurgie pancréatique. *Gastroenterol Clin Biol* 1984; 8:419-425.
 47. Warshaw AL. Conservation of pancreatic tissue by combined gastric, biliary and pancreatic duct drainage for pain from chronic pancreatitis. *Am J Surg* 1985; 149:563-569.
 48. Kerremans R, Penninckx F, De Groote J, et al. Subtotal pancreatic head resection combined with left pancreatic duct obstruction in chronic pancreatitis. *Acta Gastroenterol Belg* 1985; 48:603-606.
 49. Lippert H, Wolff H, Lorenz D, et al. Erfahrungen mit der Pankreasokklusion nach kephaler Duodenopancreatektomie. *Zentralbl Chir* 1984; 109:1112-1121.
 50. Gebhardt Ch, Stolte M. Pankreasgang-Okklusion durch Injektion einer schnellhärtenden Aminosäurenlösung. *Langenbecks Arch Chir* 1978; 346:149-166.
 51. Gooszen HG, Bosman FT, van Schilfgaarde R. The effect of duct obliteration on the histology and endocrine function of the canine pancreas. *Transplantation* 1984; 38:13-17.
 52. Gooszen HG, van Schilfgaarde R, Fröhlich M, van der Burg MPM. The effects of duct obliteration and of autotransplantation on the endocrine function of canine pancreatic segments. *Diabetes* 1985; 34:1008-1013.